study etanercept was discontinued due to primarily or secondary inefficacy (9.2%), new onset of uveitis (5.2%) and other reasons (4.0%), including injecting site reaction. No serious adverse events, including severe infections, required hospitalisation were observed during the study.

Conclusions: Etanercept enables of induction of remission in JIA patients less than 10 years, with JIA duration less than 2.4 years, and whome have HLA B27-antigene. More stable effect was observed in oligoarticular JIA patients and who received concomitant methotrexate treatment.

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AB1109

MISSED CASES OF MACROPHAGE ACTIVATION SYNDROME IN JUVENILE IDIOPATHIC ARTHRITIS (RETROSPECTIVE STUDY)

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Background: Macrophage activation syndrome (MAS) is a complication of JIA which elevates morbidity and mortality. Early recognition, differentiation from activity of JIA itself and the subsequent introduction of aggressive treatment contributes to better prognosis.

Objectives: A retrograde study to investigate the occurrence of missed MAS among JIA patients to assess the extent of the problem among those patients.

Methods: The study included 100 JIA patients registered in paediatric departments over 2 years in urban areas of Cairo. All JIA subtypes where included. MAS was diagnosed according to preliminary diagnostic guidelines for MAS complicating sJIA.^{Ravelli et al., 2005} JIA patients who had history of recent infection, hepatitis or other liver disease, malignancy and pancytopenia were excluded. The hospital registry for JIA patients was scanned for medical history with special attention for records of history suggestive of MAS. Clinical examination, Laboratory investigations and radiological data. Complementary investigations were done when needed.

Results: Among the studied 100 JIA patients, 14% of patients fulfilled the criteria and were diagnosed with MAS. Mortality rate was 2% of all JIA patients and 14% of JIA cases with MAS. Regarding gender, there were 10 females (71%) and 4 males (35%). Their age ranged from 3.5 to 18 years old (median 8 years). The disease duration ranged from 0.1 to 6 years (median 1.5 years). Ferritin level was elevated in all MAS patients. Renal affection was present in 10 MAS patients (71%) in the form of elevated serum creatinine level. In contrast to uncomplicated JIA where there was no significant renal involvement.

Table 1 Frequency of clinical manifestations of MAS among the studied patients

Clinical manifestations	JIA with MAS (n=14)	JIA without MAS (n=86)
Fever	14 (100%)	2 (2.3%)
Central nervous system manifestations	3 (21%)	-
Haemorrhage	1 (7.1%)	-
Hepatomegaly	9 (64%)	3 (3.4%)
Splenomegaly	6 (42%)	1 (1.1%)
Generalised lymphadenopathy	5 (35%)	-
Cardiovascular system manifestations	1 (7.1%)	7 (8.1%)

Table 2 Laboratory results among studied patients

Parameter	JIA with MAS Mean±SD	JIA without MAS
R red blood cells ($10^9/\text{mm}^3$)	3.4±0.6	4.66±0.73
Haemoglobin(gm/dl)	9.04±1.39	11.8±1.87
Platelets ($10^9/\text{mm}^3$)	124.29±85.8	299±90.053
WBCS ($10^9/\text{mm}^3$)	6361.43±3937.7	11989.6±6056.3
Aaspartate transaminase(u/l)	90.14±93.1	29.5±23.084
Creatinine (mg/dl)	2.19±0.91	0.73±0.2
International normalised ratio	2.04±0.6	1.07±0.14
Fibrinogen(mg/L)	203.5±86.02	317.86±67.39
Eerythrocyte sedimentation rate(mm/h)	28.29±13.12	17.56±2.83
C – reactive protein(mg/dl)	60.71±52.21	7.49±7.48

Conclusions: MAS is a potentially fulminant disorder whose incidence has increased due to raised manifestations awareness. It may be a complication of any JIA subtype but primarily systemic onset subtype. The diagnosis of MAS should be considered in presence of continuous fever, CNS, renal or haemorrhagic manifestation in JIA.

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AB1110

LIPID ABNORMALITIES IN CHILDREN AND ADOLESCENTS WITH JUVENILE IDIOPATHIC ARTHRITIS JIA

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Background: Atherosclerosis and its complications are known that one of the causes of lethality in rheumatic diseases. The risk of cardiovascular catastrophes increases not only with the prolongation of pathological process, but also in young patients, who are seropositive to rheumatoid factor and antinuclear antibodies in the early years of the disease.

Objectives: The study of lipid blood spectrum in patients with JIA with the definition of its clinical significance for the course of the disease was conducted.

Methods: 97 children (5–18 years) of patients with JIA were examined. The first group consisted of 38 children (39.2%) without symptoms of comorbidity, the second group, with signs of comorbidity, included 59 children (60.8%). The age of the debut of the disease in the first group was 6.3 years, and in the second group – 5.9 years. The duration of the disease at the time of the survey was 71 and 61 months. General clinical trials included the complex included autoantibodies, disease activity, drugs. Total cholesterol (TCh), triglycerides (TG), high density lipoprotein cholesterol, low-density lipoprotein cholesterol, apolipoprotein B, ApoA-I and lipoprotein-α were evaluated. The state of the blood coagulation system was also studied: fibrinogen of the blood, prothrombin index (PTI), thrombin time (TrT), active partial thrombin time, d-dimer, international normalised ratio.

Results: It was established that the parameters of the lipid blood spectrum changed in the presence of an increase in the level of fibrinogen. Patients with comorbidity had higher rates of low-density lipoprotein cholesterol (2.55 mmol/L versus 3.16 mmol/L; $p<0.03$), TG (0.99 mmol/L versus 1.04 mmol/L, $p<0.03$); the coefficient of atherogeny (3.10 mmol/L versus 3.24 mmol/L, $p<0.03$).

In the group of children with signs of comorbidity, there was a certain increase in the PTI and probable prolongation of the TrT ($p<0.03$), compared with the group without comorbid diseases. Children with JIA coagulation system parameters were also studied in a group of patients with signs of kidney damage. There is a probable increase in the PTI ($p<0.05$), which may indicate a predisposition to thrombosis.

It was found that in children with JIA with the presence of atherogenic changes in the lipid blood spectrum, there are signs of the hypercoagulation with parameters of the PTI ($89.70\pm0.74\%$ vs. $94.05\pm3.11\%$, $p<0.05$); fibrinogen (3.55 ± 0.25 vs. 3.96 ± 0.21 g/L, $p<0.05$) and the level of d-dimer (0.25 ± 0.08 units versus 0.82 ± 0.13 units), $p<0.01$).

Thus, in children with JIA, with signs of comorbidity, signs of hypercoagulation are established, especially in the presence of violations of the lipid profile of the blood. Regression analysis has shown that the greatest interaction exists between atherogenic dyslipidemia (the coefficient of atherogenicity) and d-dimer.

Conclusions: The obtained results confirm that the formation of comorbid pathology in children with JIA takes place against the backdrop of a close relationship between atherogenic changes in the lipid blood spectrum (the coefficient of atherogenicity) and signs of hypercoagulation (TG, d-dimer).

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